NOTES ON TWO CASES OF URETERAL ABNORMALITY.

By George Blumer, M. D.,
Assistant in Pathology, Johns Hopkins University.

[*From the Pathological Laboratory of the Johns Hopkins University and Hospital.*]

[From *The Johns Hopkins Hospital Bulletin*, Nos. 66-67, September-October, 1896.]

[From *The Johns Hopkins Hospital Bulletin*, Nos. 66-67, September-October, 1896.]

NOTES ON TWO CASES OF URETERAL ABNORMALITY.

By George Blumer, M. D.,
Assistant in Pathology, Johns Hopkins University.

[*From the Pathological Laboratory of the Johns Hopkins University and Hospital.*]

The following two cases of ureteral anomaly, which have recently come under observation, seem uncommon enough to merit description. Through the kindness of Dr. Kelly we are enabled to supplement the descriptions by plates.

The clinical histories of the cases have no special bearing upon the pathological findings, with the exception of the fact that in case No. 1 an attack of acute cystitis had occurred some five years before death; the notes are therefore confined to the pathological aspects of the cases.

Case 1.—*Anatomical Diagnosis.* Diphtheritic inflammation of the bladder, left ureter, and left renal pelvis; suppurative nephritis and perinephritis of the left kidney; hydro-ureter and hydro-nephrosis of the right kidney; miliary abscesses in the right kidney; prolapse of the ureteral and bladder mucous membrane into the bladder cavity; localized fibrinous peritonitis; acute bronchitis; fatty degeneration and cloudy swelling of the liver; slight general arterio-sclerosis.

The following is the abstract from the autopsy protocol referring to the ureters and bladder:

The right ureter is dilated to the size of a lead pencil, and contains pale, cloudy urine. The walls are thin. The mucous membrane is congested. There is nowhere any constriction until the bladder is reached.

The left ureter is dilated to about the size of the normal ileum, its walls are markedly thickened, and it contains a thick greenish black purulent material with an offensive odor. The mucous membrane has a dirty, sloughy appearance and a green-black color, its surface shows numerous irregularities in the form of patches of grayish white false membrane formation, the membrane being firmly attached to the subjacent tissue.

The bladder is somewhat enlarged and contains turbid, foul-smelling urine. Its walls are greatly thickened, measuring as much as three centimeters in their thickest part. The mucous membrane is corrugated and of a greenish black color, and shows numerous areas of densely adherent grayish false membrane similar to those seen in the ureter. Projecting into the bladder from a point where the left ureteral orifice is normally present is a pyramidal sac, tense and fluctuating, and evidently containing fluid. This sac hangs free in the cavity of the bladder and reaches from its place of origin nearly to the internal urethral orifice. It is about eight centimeters in length, has a narrow neck, measuring three centimeters in diameter where it joins the bladder wall, and gradually expands as it passes out from this point, reaching its greatest diameter of nine centimeters a short distance from its free end. On its inner side, about its middle, a minute opening can be made out. This opening is circular and just about large enough to admit a pin point; it is situated in the center of a small area of dense fibrous tissue, and is evidently the lower opening of the left ureter. On opening the sac it is found to contain a thick greenish black purulent material similar to that already seen in the left ureter, and the index finger can be passed directly from the sac into the dilated ureter. Both the internal and external surfaces of the sac are covered with mucous membrane which shows patches of false membrane formation similar to those seen in the ureter and bladder. The sac wall appears to be of the same thickness throughout.

From the normal site of the right lower ureteral orifice there projects a similar but much smaller sac three centimeters in length; it is not nearly so tense as the one on the other side. On the inner side of this second sac is an opening the size of a pin's head, from which urine escapes; there is apparently no cicatricial tissue about this opening. The sac communicates freely with the left ureter. The prostate gland is slightly enlarged, but not sufficiently so to offer any obstruction to the outflow of urine.

The urethra is free from obstruction throughout its entire course. A microscopical examination of the sac wall shows that between the two layers of mucous membrane lies con-

nective tissue containing many bundles of unstriped muscle fiber.

Remarks. This peculiar prolapse of the mucous membrane of the lower end of the ureter and that of the adjacent part of the bladder is not of common occurrence, although we find some similar cases on record.

All of these cases seem to come under two main categories:

1. Those cases in which there is distinct evidence that the condition is due to congenital deformity.

2. Those cases in which the process has apparently resulted from some acquired abnormality of the genito-urinary tract. By far the larger number of cases present distinct evidence of congenital malformation; in fact, out of the thirteen cases which we have been able to collect, ten were evidently of congenital origin, death occurring in five of these in the early years of childhood.

All of these five cases occurred in female children, and in four of them, those of Davies-Colley, Caillé, Beach, and Geerdts, there was not only a prolapse into the bladder, but the prolapsed sac actually passed through the urethra and appeared externally.

In the congenital cases proving fatal at a later date, the deformity was in some instances less severe than in the cases ending fatally in early life; in other instances the deformity was just as great in the late as in the early cases, the previous escape of the individual perhaps being due to the fact that he had escaped genito-urinary infection, which seemed to have been the cause of death in the majority of the cases which succumbed early in life. The deformity in these congenital cases consisted in most instances of a partial or complete closure of the lower ureteral orifice. In one case another form of deformity is cited, viz. a ureter having a long portion of its course in the bladder wall, and for this reason subjected to an abnormal amount of pressure from the bladder musculature.

Accompanying the deformity which was the actual cause of the condition, were often other evidences of congenital malformation. In several instances abnormalities of the kidney pelvis or double ureters were present, and one case is cited in which a deformity of the uterus was noted.

Of the cases in which there was no apparent congenital cause for the condition—and to this class we consider our case belongs—we could find but two examples, one reported by T. Smith, the other by Hutinel.

Smith's case, judging from his description and from an excellent plate which illustrates his article, was almost exactly similar to our own. In his case, however, urinary calculi were present on the right side in the prolapsed pouch, and on the left in the renal pelvis.

In Hutinel's case, which occurred in an old man, the subject of cystitis, the pouching only involved the left ureter. At the time of death the lower orifice of the left ureter was not blocked, as the pouch communicated with the bladder by a pouch the size of a three-franc piece, but there was evidence of a former blocking in a much atrophied kidney on the affected side. Unfortunately Hutinel does not state whether the abnormally large opening which existed between the sac and the bladder was apparently due to simple dilatation or to an ulcerative process.

Judging from the fact that in our case the abnormally small ureteral opening was contained in an area of cicatricial tissue, and from the history that the patient gave of a previous attack of acute cystitis, we are inclined to think that the blocking of the lower ureteral opening was due to an old inflammatory process with a subsequent formation of new tissue, which, in contracting, narrowed the ureteral orifice. It would seem likely that in the cases of Smith and Hutinel the same process might have taken place, as both patients showed evidence of old inflammatory disease of the genito-urinary tract.

As far as the mechanism of the pouching is concerned the cases can again be divided into two classes, those in which there is a uniform dilatation from the pelvis of the kidney down to the end of the sac, and those in which the pelvis of the kidney and the main portion of the ureter are not dilated, the sacculation occurring only at the extreme lower end of the ureter.

In the former class of cases, to which ours belongs, the pouching can be explained by a theory, the essence of which was advanced by Smith in reporting his case. He suggests

that the prolapse in these cases is due to the disproportion between the capacity of the ureter and kidney pelvis on the one hand, and the size of the lower ureteral orifice on the other. When, either from an extreme hydro-nephrosis or from an abnormal narrowing of the lower ureteral orifice, the urine is no longer able to escape from this orifice as fast as it is secreted, the distended ureter and kidney pelvis are compressed by the action of the abdominal muscles, particularly during urination and defecation, and a considerable downward pressure is brought to bear on the lower ureteral orifice. This finally results in the prolapse of its mucous membrane, and as the mucous membrane of the bladder is directly continuous with that of the ureter, it is of course pushed downward at the same time, the result being a pouch covered on both sides by mucous membrane, and directly continuous with the dilated ureter and kidney pelvis.

In the cases in which no hydro-nephrosis or hydro-ureter exists—and most of the congenital cases are of this kind—we must seek for some further abnormality in connection with the lower end of the ureter.

According to Boström, this abnormality consists in the manner in which the ureter passes through the bladder wall.

In the normal condition, this author states, the ureters pass obliquely through the bladder wall, and their lower openings lie partly in the bladder musculature, the contraction of the muscle counteracting the downward pressure of the urine and hindering dilatation of the portion of the ureter lying within the musculature.

In the cases in which sacculation takes place the ureter is stated to pass straight through the bladder wall and to end just beneath the mucous membrane, in those cases where the ending is blind. From this it results, according to Boström, that the area of ureter surrounded by muscle is much smaller than normal, the resistance to the downward pressure of the urine is insufficient, and a prolapse of the mucous membrane of the ureter and bladder results. That the portion of the bladder wall which surrounds the ureter is not included in the prolapsed tissue is proved, Boström thinks, by the absence of muscular tissue from the wall of the sac.

Burckhard, in his paper, modifies somewhat Boström's

theory, holding that the primary cause of the abnormality lies not so much in the abnormal position of the ureter as in the congenital lack of musculature in the bladder wall. He also states that the dilated portion of the ureter does not originate, as Boström believes, in the portion just beneath the mucous membrane of the bladder, but on the contrary begins in the portion which lies within the bladder wall, the proof of this lying in the fact that in his case he was able to demonstrate unstriped muscle fiber in the wall of the sac. The absence of muscle fiber in the sac wall in Boström's case he explains on the grounds of disappearance from pressure atrophy.

Case 2.—*Anatomical Diagnosis.* Sloughing carcinoma of the uterus involving the vagina and bladder; involvement of both ureters in adhesions; hydro-nephrosis and hydro-ureter; kinking of the right ureter from the passage of the right ovarian vein over the dilated ureter; arterio-sclerosis; heart hypertrophy; recent vegetative endocarditis; emphysema of the lungs; diverticulæ of the intestine.

The following is the abstract from the autopsy protocol (Dr. Flexner) referring to the ureter:

The ureter on the right side is greatly dilated. The greatest dilatation is in the upper third, next the hilum of the kidney. At its entrance into the kidney there is a pyriform bulging of the ureter, the walls of which are so attenuated at this point as to permit of the slightly turbid but almost colorless contents being seen through them. The ureter just next to the hilum, and corresponding with the smaller part of the pyriform enlargement, has the size of a thumb. At a distance of five centimeters from the hilum of the kidney a sharp bend occurs in the ureter, which is much constricted at this point by the passage over it of the ovarian vein, the walls of which are here thickened, but the lumen not entirely occluded. It is owing to the pressure from this vein that the upper part of the ureter is so much more dilated than the lower part, which will be described later. The pressure from above has caused the pushing downward of the upper portion of the dilated ureter, thus partly covering over the lower portion and causing an S-shaped bend or curve.

The lower portion of the ureter has a length of ten to twelve

centimeters, and at the uterine end is embedded in firm adhesions. The dilatation of this part is somewhat less than that of the upper portion, and on an average it is about the size of the index finger. The lower part of the ureter contains the same faintly turbid fluid contained in the upper part. A probe cannot be passed from the slit up ureter into the bladder, even with the use of a moderate amount of force.

Remarks. In this case the hydro-ureter and hydro-nephrosis were evidently due in the first case to the inclusion of the lower end of the ureter in adhesions. The peculiar kinking, which was the interesting feature of the case, was due to the passage across the dilated ureter of what appeared to be the right ovarian vein, though instead of emptying into the inferior vena cava, as would normally be the case, it terminated in the right renal vein. As the vein was not in any way bound down, its mere tautness was evidently sufficient to cause compression of the dilated ureter.

We have been unable to find an account of a similar case.

Bibliography.

Beach: Transactions of the Pathological Society, London, 1873–4, vol. XXV, p. 185.

Boström: Beiträge zur pathologischen Anatomie der Nieren. Freiburg and Tübingen, 1884.

Burckhard: Centralblatt für allgemeine Pathologie und pathologische Anatomie, Bd. VII, No. 4, p. 129.

Caillé: American Journal of the Medical Sciences, vol. 95 N. S., p. 481.

Davies-Colley: Lancet, London, March 15, 1879.

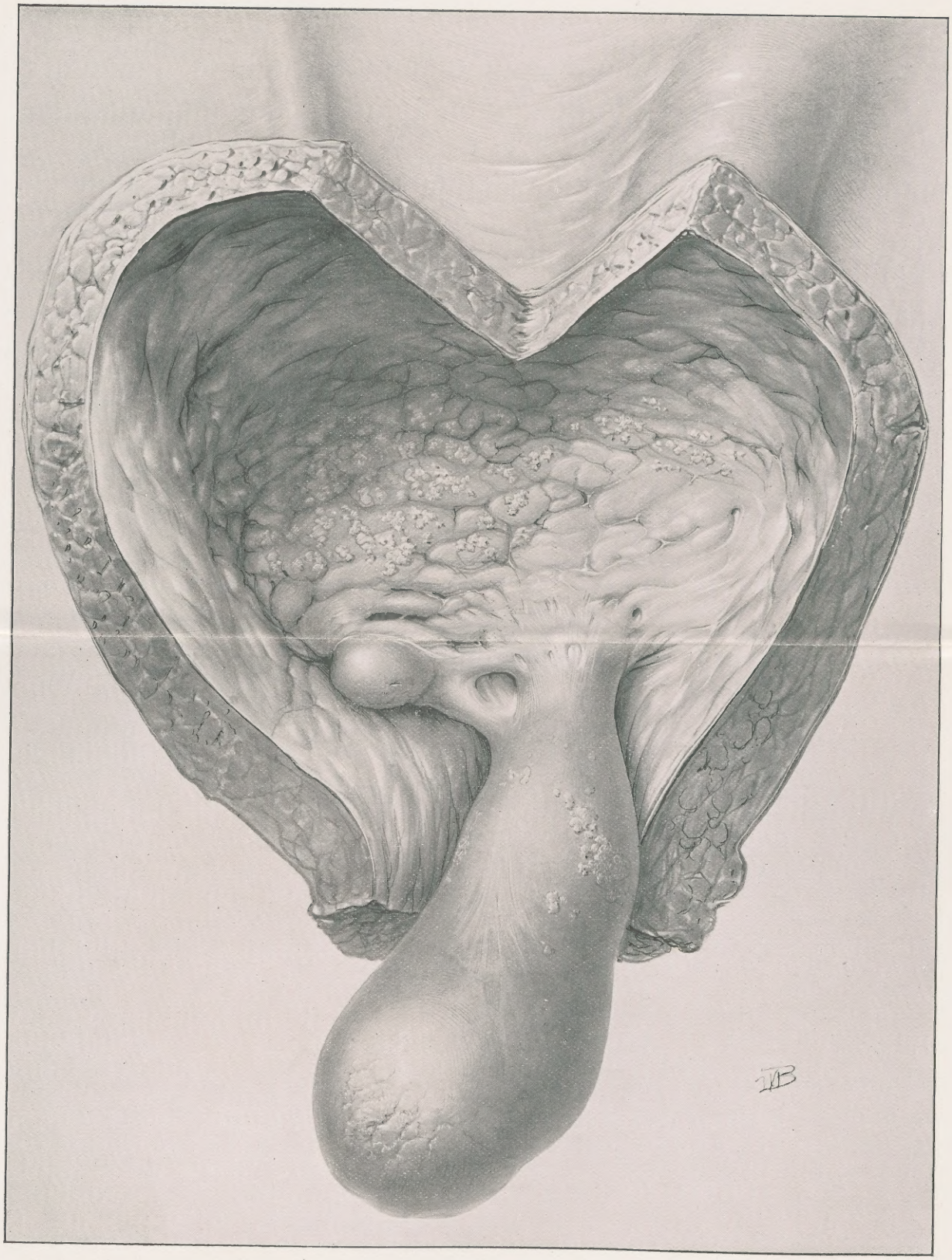
Geerdts: cited by Schwartz.

Hutinel: Bulletin de la Société d'Anatomie de Paris, 1873, vol. XLVIII, p. 695.

v. Lechler-Neelsen: cited by Schwartz.

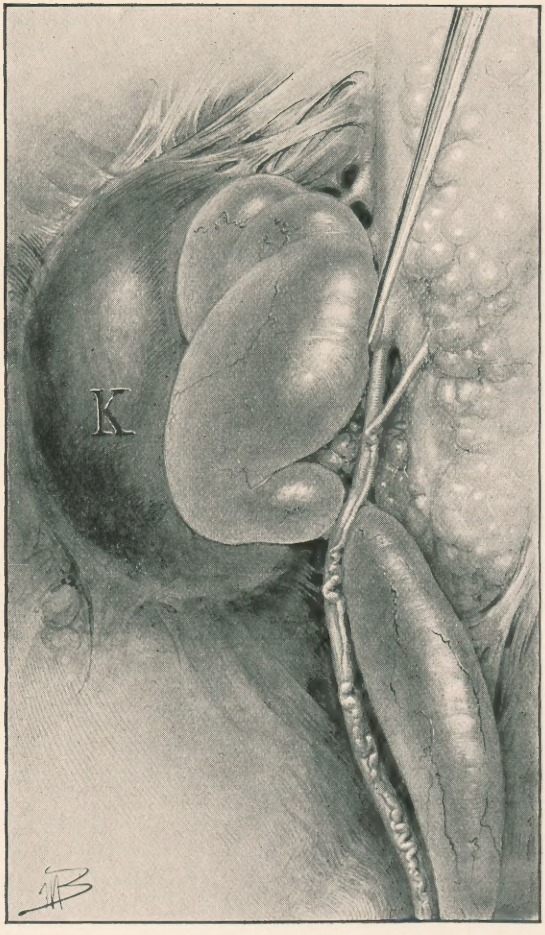
Schwartz: Beiträge zur klinischen Chirurgie, Bd. XV, Hft. 1, 1895.

Tange: Virchow's Archiv, Bd. 118, 1889, p. 414.



CASE I.

The Friedenwald Co., Engravers and Printers.



CASE II.

The Friedenwald Co., Engravers and Printers.